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Long term relationship of mean arterial pressure

In the cardiovascular system, blood flow is controlled by arterial blood pressure, and in this way the long-term mean blood pressure is stabilized to regulate oxygen and carbon dioxide levels. Thereafter, the baroreflex would stabilize the instantaneous **pressure** value to the prevailing carotid pressure.

There are several physiological mechanisms that regulate blood pressure in the long-term, the first of which is the renin-angiotensin-aldosterone system (**RAAS**).

Renin-Angiotensin-Aldosterone System (RAAS)

Renin is a peptide hormone released by the granular cells of the **juxtaglomerular apparatus** in the kidney. It is released in response to:

Sympathetic stimulation

Reduced sodium-chloride delivery to the distal convoluted tubule

Decreased blood flow to the kidney

Renin facilitates the conversion of angiotensinogen to angiotensin I which is then converted to angiotensin II using angiotensin-converting enzyme (**ACE**).

Angiotensin II is a potent vasoconstrictor. It acts directly on the kidney to increase sodium reabsorption in the proximal convoluted tubule. Sodium is reabsorbed via the sodium-hydrogen exchanger. *Angiotensin II* also promotes release of **aldosterone**.

ACE also breaks down a substance called **bradykinin** which is a potent vasodilator. Therefore, the breakdown of bradykinin potentiates the overall constricting effect.

Aldosterone promotes salt and water retention by acting at the distal convoluted tubule to increase expression of **epithelial** sodium channels. Furthermore, aldosterone increases the activity of the basolateral sodium-potassium ATP-ase, thus increasing the electrochemical gradient for movement of sodium ions.

More sodium collects in the kidney tissue and water then follows by osmosis. This results in decreased water excretion and therefore increased blood volume and thus blood pressure.

Pulmonary circulation:

Pulmonary circulation, system of **blood** vessels that forms a closed circuit between the **heart** and the **lungs**, as distinguished from the systemic circulation between the heart and all other body tissues. On the evolutionary cycle, pulmonary circulation first occurs in **lungfishes** and **amphibians**, the first

animals to acquire a three-chambered heart. The pulmonary circulation becomes totally separate in crocodilians, [birds](#), and [mammals](#), when the ventricle is divided into two chambers, producing a four-chambered heart. In these forms the pulmonary circuit begins with the right ventricle, which pumps deoxygenated blood through the [pulmonary artery](#). This artery divides above the heart into two branches, to the right and left lungs, where the arteries further subdivide into smaller and smaller branches until the capillaries in the pulmonary air sacs (alveoli) are reached. In the capillaries the blood takes up [oxygen](#) from the air breathed into the air sacs and releases [carbon dioxide](#). It then flows into larger and larger vessels until the [pulmonary veins](#) (usually four in number, each serving a whole lobe of the lung) are reached. The pulmonary veins open into the left atrium of the heart.

Circle of willis:

The circle of Willis encircles the stalk of the pituitary gland and provides important communications between the blood supply of the forebrain and hindbrain (ie, between the internal carotid and [vertebro-basilar](#) systems following obliteration of primitive embryonic connections). ^[1] Although a complete circle of Willis is present in some individuals, it is rarely seen radiographically in its entirety; anatomical variations are very common and a well-developed communication between each of its parts is identified in less than half of the population. ^[1]

The circle of Willis begins to form when the right and left internal carotid artery (ICA) enters the cranial cavity and each one divides into two main branches: the anterior cerebral artery (ACA) and [middle cerebral artery](#) (MCA). ^[2] The anterior cerebral arteries are then united and blood can cross flow by the anterior communicating (ACOM) artery. The ACAs supply most midline portions of the frontal lobes and superior medial parietal lobes. The MCAs supply most of the lateral surface of the hemisphere, except the superior portion of the parietal lobe (via ACA) and the inferior portion of the temporal lobe and occipital lobe. The ACAs, ACOM, and MCAs form the anterior half, better known as the anterior cerebral circulation. Posteriorly, the basilar artery (BA), formed by the left and right vertebral arteries, branches into a left and right [posterior cerebral artery](#) (PCA), forming the posterior circulation. ^[3] The PCAs mostly supply blood to the occipital lobe and inferior portion of the temporal lobe

4. Splanchnic circulation:

The splanchnic circulation is composed of gastric, small intestinal, colonic, pancreatic, hepatic, and splenic circulations, arranged in parallel with one another. The three major arteries that supply the splanchnic organs, celiac and superior and inferior mesenteric, give rise to smaller arteries that anastomose extensively. The circulation of some splanchnic organs is complicated by the existence of an intramural circulation. Redistribution of total blood flow between intramural vascular circuits may be as important as total blood flow. Numerous extrinsic and intrinsic factors influence the splanchnic circulation. Extrinsic factors include general hemodynamic conditions of the cardiovascular system, autonomic nervous system, and circulating neurohumoral agents. Intrinsic mechanisms include special properties of the vasculature, local metabolites, intrinsic nerves, paracrine substances, and local hormones. The existence of a multiplicity of regulatory mechanisms provides overlapping controls and restricts radical changes in tissue perfusion.

5. Coronary circulation:

Coronary circulation is the circulation of blood in the blood vessels that supply the heart muscle (myocardium). Coronary arteries supply oxygenated blood to the heart muscle, and cardiac veins drain away the blood once it has been deoxygenated. Because the rest of the body, and most especially the brain, needs a steady supply of oxygenated blood that is free of all but the slightest interruptions, the heart is required to function continuously. Therefore its circulation is

of major importance not only to its own tissues but to the entire body and even the level of consciousness of the brain from moment to moment. Interruptions of coronary circulation quickly cause heart attacks (myocardial infarctions), in which the heart muscle is damaged by oxygen starvation. Such interruptions are usually caused by ischemic heart disease (coronary artery disease) and sometimes by embolism from other causes like obstruction in blood flow through vessels.

6. Cutaneous circulation:

The cutaneous circulation is the circulation and blood supply of the skin. The skin is not a very metabolically active tissue and has relatively small energy requirements, so its blood supply is different to that of other tissues.

Some of the circulating blood volume in the skin will flow through will flow through **arteriovenous anastomoses (AVAs)** instead of capillaries. AVAs serve a role in temperature regulation. In this article we shall consider the different adaptations of the cutaneous circulation, and its role in body temperature control.

7. Cardiovascular adjustment during exercise:

The integrated response to severe exercise involves fourfold to fivefold increases in cardiac output, which are due primarily to increases in cardiac rate and to a lesser extent to augmentation of stroke volume. The increase in stroke volume is partly due to an increase in end-diastolic cardiac size (Frank-Starling mechanism) and secondarily due to a reduction in end-systolic cardiac size. The full role of the Frank-Starling mechanism is masked by the concomitant tachycardia. The reduction in end-systolic dimensions can be related to increased contractility, mediated by beta adrenergic stimulation. Beta adrenergic blockade prevents the inotropic response, the decrease in end-systolic dimensions, and approximately 50% of the tachycardia of exercise.

The enhanced cardiac output is distributed preferentially to the exercising muscles including the heart. Blood flow to the heart increases four-fold to fivefold as well, mainly reflecting the augmented metabolic requirements of the myocardium due to near maximal increases in cardiac rate and contractility. Blood flow to the inactive viscera (e.g., kidney and gastrointestinal tract) is maintained during severe exercise in the normal dog. It is suggested that local autoregulatory mechanisms are responsible for maintained visceral flow in the face of neural and hormonal autonomic drive, which acts to constrict renal and mesenteric vessels and to reduce blood flow. However, in the presence of circulatory impairment, where oxygen delivery to the exercising muscles is impaired as occurs to complete heart block where normal heart rate increases during exercise are prevented, or in congestive right heart failure, where normal stroke volume increases during exercise are impaired, or in the presence of severe anemia, where oxygen-carrying capacity of the blood is limited, visceral blood flows are reduced drastically and blood is diverted to the exercising musculature. Thus, visceral flow is normally maintained during severe exercise as long as all other compensatory mechanisms remain intact. However, when any other compensatory mechanism is disrupted (even the elimination of splenic reserve in the dog), reduction and diversion of visceral flow occur.